Thomas in 1902 found that the ganglia are often involved. The peripheral terminations of these cells would then degenerate and present peripheral neuritis. In regard to tabetic degenerations, though he does not explain why it begins there, Nageotte yet shows that when a nerve fiber is attacked that the decay tends to begin at the extreme periphery just as it commences there in a decaying tree. Dr. Williams said that any little novelty he brought to the discussion laid in the facts he presented as to the Obersteiner ring. The process itself begins, as Nageotte says, in the root, but in early cases the first indication is often seen at the radicular ring, because there the nerve loses its neurilemma sheath. One might fail to notice the fact that in the root some fibers are missing, whereas the degeneration beyond the radicular ring persists and is manifest to the older methods of examination. Careful examination shows always degeneration of the posterior root, but much more marked degeneration of the spinal side of the radicular ring. The reason for that is that the nerves have lost their neurilemma sheath there. Only careful examination will show that there are in the roots regenerating fibers. The regeneration is such as to give rise to early resumption of activity in the muscles, so that it is often clinically unappreciable except by careful examination. Another point cogent in Dr. Spiller's remarks was that he had so often noted muscular atrophy. He has observed frequency of muscular atrophy in tabetics. He has also observed the frequency of meningitis. The alleged absence of muscular atrophy clinically and meningitis postmortem were two weak points in adhering to the doctrine of Nageotte. Dr. Spiller's cooperation in the removal of these objections is most gratifying to Dr. Williams, who said he was very glad indeed to have had the opportunity to present his paper before the Philadelphia Neurological Society and thanked the society very much for the manner in which his paper had been received.

THE AMERICAN NEUROLOGICAL ASSOCIATION

The Thirty-fourth annual meeting held at the College of Physicians of Philadelphia, May 20, 21 and 22, 1908

The President, Dr. Charles W. Burr, in the Chair.

The President delivered his address, entitled: The Mental State in Chorea and Choreiform Affections. (See this journal, p. 353.)

PROGRESSIVE HEMIPLEGIA DUE TO GLIOSIS AND VASCULAR LESION OF THE RIGHT CENTRUM AND CORTEX

By Frank R. Fry, M.D.

Eight weeks before the necropsy there were slight recurring spasm and paresthesia of the face, and a still slighter spasmodic tendency in the hand on the left side. There was a partial paralysis of the hand and arm which had been gradually advancing for four months. There was no paralysis in the lower extremity. There was a complete absence of objective sensory signs in the paralytic member and elsewhere. There were also absent the general signs of cerebral tumor, *i. e.,* no vertigo,

vomiting, headache, or optic neuritis. The diagnosis was a lesion, probably a neoplasm, in the ascending frontal convolution. The patient later developed general convulsions and a full hemiplegia of the left side.

He died after an operation. A gliosis was found which invaded much of the frontal lobe and backwards slightly beyond the ascending parietal convolution.

The features of the case were: the presence of paresthesia or subjective sensory phenomena for many weeks before objective signs of sensory disturbance appeared in the same areas; the diagnosis of a probable tumor from the presence of an advancing paralysis with symptoms of corresponding cortical irritation, although all the general symptoms of tumor were absent; the presence of data from which an extensive growth of the glosis in seven to eight weeks could be estimated.

A CASE OF APRAXIA WITH AUTOPSY.

By John H. W. Rhein, M.D.

(To be published in this journal.)

Man of fifty-five developed progressive loss of eyesight, together with inability to recognize the position of his body and limbs; loss of the sense of location generally; loss of tactile sense in the left hand, and disturbance of the temperature sense in the left hand. The stereognostic perception was impaired on both sides. Finally, there was a contracture of the left arm and hand, with rigidity of the left leg, some rigidity of the right arm and leg, but not to the point of contracture. Progressive mental failure.

Autopsy revealed an area of softening in the cortex, in the occipito-parietal region on the right side. Sections of the brain showed degeneration in the white matter of both occipital lobes, and part of the parietal lobes.

Discussion of apraxia.

Discussion.

Dr. Joseph Collins said that the case reported had very decided interest for him because the patient had been for a brief time under his observation and because he had utilized him both at the American Neurological Association meeting in Boston two years ago and at the meeting of the New York and Philadelphia Neurological Societies in this city a year or so ago, as an illustration of a clinical type of cerebral arteriosclerosis which he has frequently encountered and to which he had desired to call attention. This patient was considered by some physicians who had observed him to have a brain lesion of the nature of a neoplasm. When Dr. Collins saw him he was receiving antisyphilitic medication. Dr. Collins' diagnosis was made in regard to his lesion, entirely aside from his agnosia, apraxia, and loss of the sense of position which conditions he had then much as Dr. Rhein related them to-day. Dr. Collins maintained then and later that the patient conformed in practically all the clinical manifestations with a type of arteriosclerosis which he had described at the meetings mentioned and he thinks that the autopsy corroborates that view. So far as Dr. Collins could judge from the brief report which was given by Dr. Rhein the morbid condition was in reality a softening, probably dependent upon a widespread arteriosclerosis,